

Allergy and Asthma : Contents

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Q: Enumerate the chemical mediators of allergic reactions and describe the important actions of histamine

Chemical Mediators of Allergic Reactions

Preformed Mediators

1. **Histamine**: Stored in mast cells and basophils; released immediately upon allergen exposure.
2. **Tryptase**: A serine protease found in mast cells.
3. **Chymase**: Another serine protease stored in mast cells.
4. **Heparin**: An anticoagulant stored in mast cells.
5. **Platelet-Activating Factor (PAF)**: Involved in platelet aggregation and degranulation.

Newly Synthesized Mediators

1. **Leukotrienes (C4, D4, E4)**: Potent bronchoconstrictors and increase vascular permeability.
2. **Prostaglandins (D2, F2 α)**: Involved in bronchoconstriction and vasodilation.
3. **Thromboxanes**: Involved in platelet aggregation and bronchoconstriction.
4. **Cytokines (IL-4, IL-5, IL-13)**: Involved in immune cell recruitment and activation.
5. **Chemokines (MCP-1, MIP-1)**: Chemotactic factors for immune cells.

Enzymes

1. **Phospholipase A2**: Catalyzes the release of arachidonic acid, a precursor for prostaglandins and leukotrienes.
2. **Cyclooxygenase (COX)**: Involved in the synthesis of prostaglandins.
3. **Lipoxygenase**: Involved in the synthesis of leukotrienes.

Others

1. **Nitric Oxide (NO)**: A gaseous mediator involved in vasodilation.
2. **Reactive Oxygen Species (ROS)**: Involved in tissue damage and inflammation.

Important Actions of Histamine

1. **Vasodilation**: Histamine acts on H1 receptors to cause dilation of arterioles, leading to increased blood flow to the affected area.

2. **Increased Vascular Permeability:** It causes the endothelial cells of venules to contract, leading to gaps through which plasma and immune cells can pass.
3. **Bronchoconstriction:** Histamine release in the lungs can lead to bronchial smooth muscle contraction, manifesting as wheezing or difficulty in breathing.
4. **Gastric Acid Secretion:** Histamine stimulates H₂ receptors on parietal cells in the stomach to increase gastric acid secretion.
5. **Pruritus:** Activation of H₁ receptors on sensory nerve endings leads to itching.
6. **Urticaria:** Histamine release is a common cause of hives or skin rash.
7. **Mucous Secretion:** It stimulates the glands to produce more mucus, contributing to symptoms like a runny nose.
8. **Cardiac Effects:** Although less common, histamine can affect the heart rate and contractility.
9. **Neurotransmission:** Histamine acts as a neurotransmitter in the brain, affecting sleep and cognitive functions.

Allergic tendency and process:

1. **Altered Reactivity in Allergic Patients:** Individuals with allergies exhibit heightened sensitivity to environmental and food antigens that are otherwise innocuous to non-allergic individuals. This heightened sensitivity is often mediated by Immunoglobulin E (IgE) antibodies.
2. **Clinical Manifestations and Familial Predisposition:** Allergic reactions manifest as hyperresponsiveness in target organs like the lungs, skin, gastrointestinal tract, and nose. These conditions often have a familial predisposition.
3. **Environmental Factors:** The surge in allergic diseases is attributed to various environmental factors such as air pollution, tobacco smoke, respiratory viruses, and possibly a decline in certain infectious diseases (hygiene hypothesis).
4. **Allergens and Biochemical Properties:** Allergens are primarily proteins that induce IgE production, leading to a type 1 hypersensitivity response. Factors like the allergen's biochemical properties, stability, and dose contribute to its allergenic potential.
5. **Molecular Weight of Allergens:** Most allergens have a molecular weight ranging from 10-70 kDa. Molecules outside this range are less likely to induce allergic reactions.
6. **T-Cell Response:** Atopic individuals exhibit a rapid expansion of T-helper type 2 (Th₂) cells that secrete cytokines favoring IgE synthesis and eosinophilia.

7. **Th2 Cytokines:** IL-4 and IL-13 are crucial for immunoglobulin isotype switching to IgE. IL-5 and IL-9 are important for eosinophil differentiation and development.
8. **IL-25, IL-33, and TSLP:** These cytokines secreted from epithelial cells contribute to Th2 response and eosinophilia.
9. **Th1 Cells and Chronicity:** Th1 cells contribute to the chronicity and effector phase in allergic diseases, inducing activation and apoptosis of epithelial cells.
10. **Th17 and Th22 Cells:** These cells link the immune response to tissue inflammation. IL-17 augments inflammation, whereas IL-22 plays a tissue-protective role.
11. **T-Regulatory Cells (Tregs):** These cells suppress effector T cells and are critical in the expression of allergic and autoimmune diseases. They express CD4+CD25+ surface molecules and immunosuppressive cytokines like IL-10 and TGF- β 1.
12. **Innate Lymphoid Cells (ILCs):** These cells lack rearranged T- and B-cell antigen receptors but show similarities to Th1, Th2, and Th17/Th22 types of immune responses. They play roles in protection against infection and the pathogenesis of inflammatory diseases.
13. **Antigen-Presenting Cells (APCs):** Dendritic cells, Langerhans cells, monocytes, and macrophages present allergens to T cells and modulate allergic inflammation.
14. **IgE and Its Receptors:** The acute allergic response depends on IgE and its ability to bind selectively to Fc ϵ RI or Fc ϵ RII. Cross-linking of receptor-bound IgE molecules initiates allergic inflammation.
15. **Eosinophils:** Characterized by peripheral blood and tissue eosinophilia, they participate in both innate and adaptive immune responses. Eosinophils are a rich source of prostaglandins and leukotrienes.
16. **Mast Cells:** Derived from CD34 hematopoietic progenitor cells, they are widely distributed throughout connective tissues. They contain a diverse array of mediators affecting allergic inflammation and organ function.
17. **Chemokines:** These are chemotactic cytokines that play a central role in tissue-directed migration of inflammatory cells. RANTES, MIP-1 α , and eotaxins are important for recruiting eosinophils into local allergic tissue inflammatory reactions.

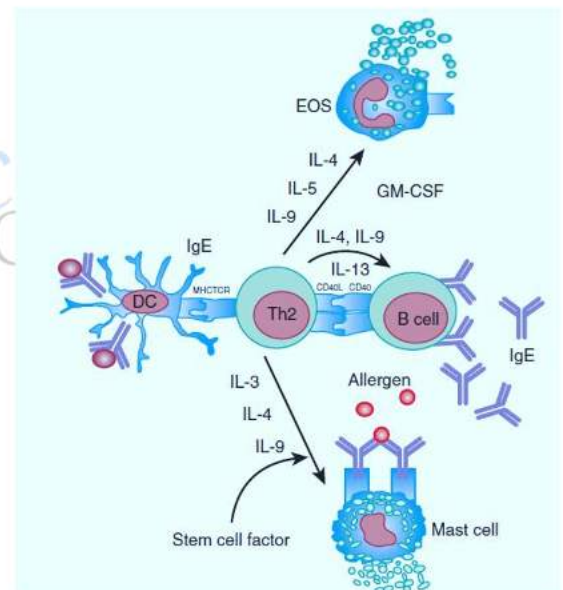
18. **Chronic Allergic Disease:** Persistent tissue inflammation can last from days to years, often due to recurrent exposure to allergens and microbial agents. Tissue remodeling leading to irreversible changes in target organs is also a feature of chronic allergic disease.
19. **Type 2 Immune Response:** Generally underlines a majority of asthma cases, atopic dermatitis, chronic rhinosinusitis, and allergic rhinitis. It involves Th2 cells, type 2 B cells, ILC2, IL-4 secreting NK T cells, basophils, eosinophils, and mast cells.
20. **Complex Network of Cytokines:** IL-4, IL-5, IL-9, and IL-13 are mainly secreted from the immune system cells, and IL-25, IL-31, IL-33, and TSLP from tissue cells, particularly epithelial cells.

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Q: Role of T cell subsets in allergic cascade

Role of Th2 cytokines in allergic cascade

- **Th2 Cells:** Central role in allergic reactions, producing specific cytokines.
- **EOS (Eosinophils):** Activated by several cytokines such as IL-4, IL-5, and GM-CSF.
- **DC (Dendritic cells):** Crucial for antigen presentation, interact with Th2 cells.
- **B Cells:** Produce IgE in response to allergens, influenced by cytokines like IL-4, IL-9, and IL-13.
- **Mast Cells:** Activated by allergens binding to IgE; release various allergic mediators.
- **Allergens:** External agents that trigger allergic reactions.
- **Cytokines:** Play roles in mediating allergic responses; including IL-3, IL-4, IL-5, IL-9, and IL-13.

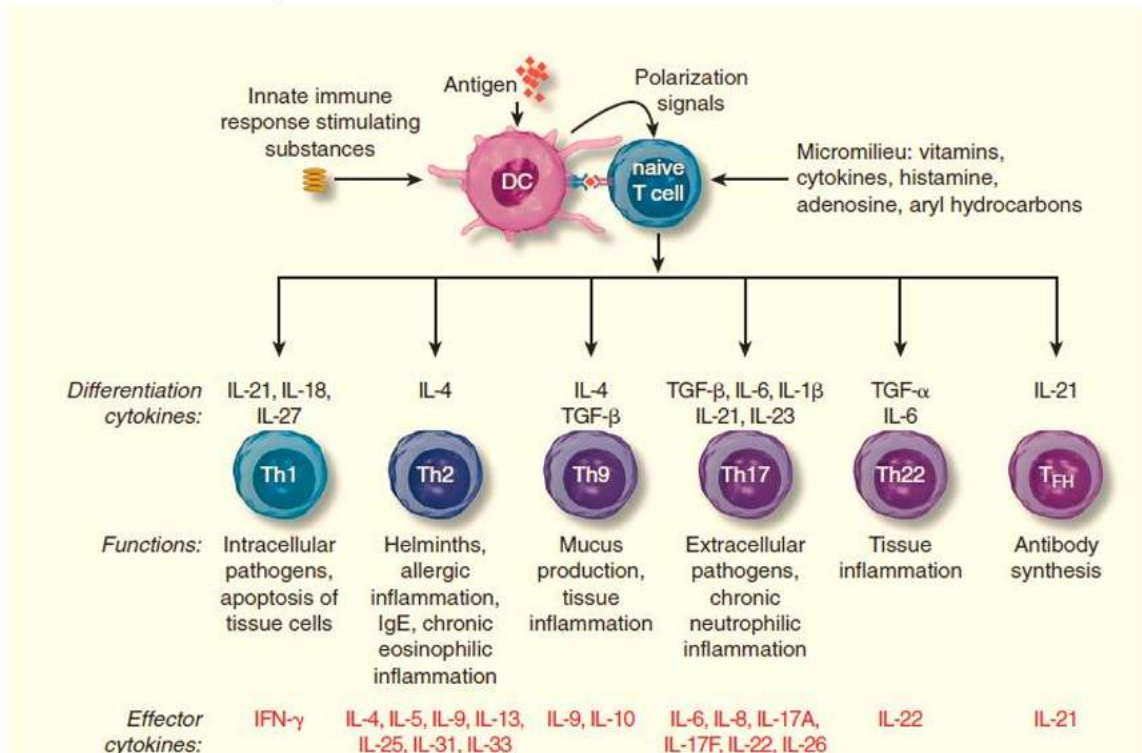


IMP Points:

- Th2 cells and their cytokines play central roles in the allergic cascade.
- IL-4, IL-9, and IL-13 are crucial cytokines that mediate the allergic response.
- Allergens initiate complex interactions involving various immune cells and cytokines.

Effector T-cell subsets

- **Differentiation Cytokines:** Several cytokines, such as IL-21, IL-18, and IL-27, play a role in T-cell differentiation into various subsets.
- **Th1, Th2, Th9, Th17, Th22, and Tfh:** Different T-cell subsets, each with specific roles in immune responses.



- **Functions:** Each T-cell subset has a unique role. For instance,
 - Th1 cells are involved in intracellular pathogen defense.
 - Th2 cells deal with helminth infections and allergic reactions.
 - Th9 cells are associated with mucus production and tissue inflammation.
 - Th17 cells deal with extracellular pathogens.
 - Th22 cells are linked to tissue inflammation.
 - Tfh cells aid in antibody synthesis.

IMP Points:

- Dendritic cells (DCs) present antigens to naïve T cells, leading to differentiation.

- Differentiation into Th2 is associated with allergic responses.
- Micromilieu, including cytokines, vitamins, histamines, adenosine, and more, plays a role in T-cell differentiation.

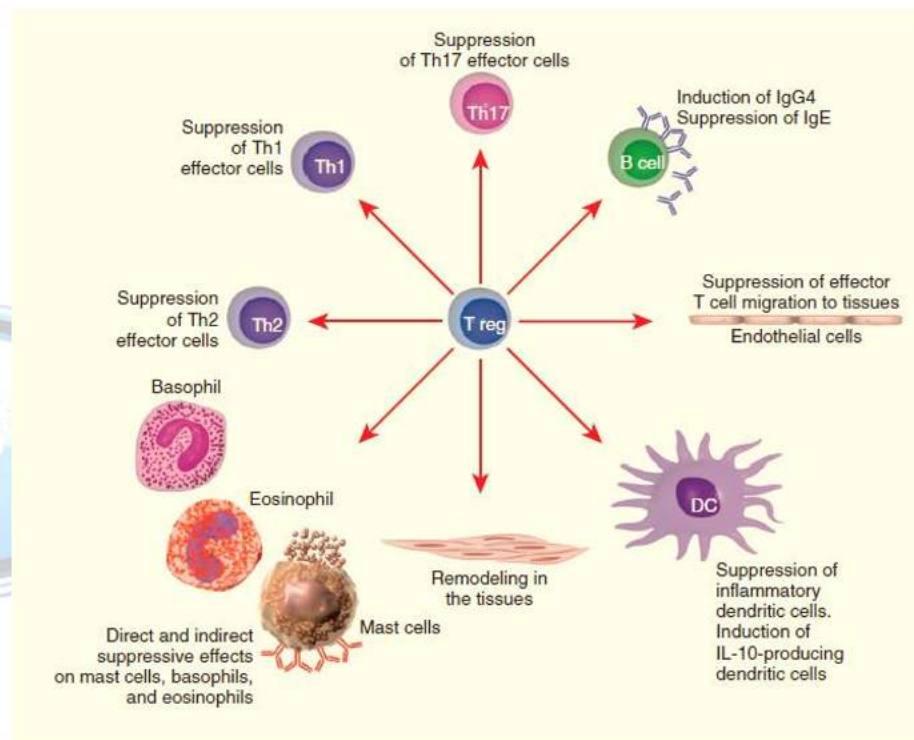
Control of allergen-specific immune responses

- **Treg (Regulatory T cells):** Central to suppressing various effector T-cell types.
- **Suppression:** Various mechanisms suppress the effect of Th1, Th2, and Th17 cells.

- **DC (Dendritic cells):** Play a central role in antigen presentation and subsequent T-cell responses.

- **Other Cells:** Eosinophils, basophils, mast cells, and endothelial cells are involved in allergic reactions.

- **Remodeling in tissues:** Result of allergic reactions in affected tissues.



- **Induction and Suppression:**
 - Induction of IgG4.
 - Suppression of IgE and other T-cell responses.

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Q: Allergic Rhinitis

- Allergic rhinitis (AR) is a prevalent condition, impacting approximately 20–30% of children.

- ✦ It manifests as an inflammatory disorder of the nasal mucosa, primarily characterized by nasal congestion, rhinorrhea, and itching. Sneezing and conjunctival inflammation can also be observed.
- ✦ AR stands out as a significant medical condition due to its considerable impact on quality of life, school attendance, and job performance.
- ✦ If left untreated, AR can result in more severe conditions like otitis media, sinusitis, and sleep disorders.
- **Symptom Duration:**
 - Intermittent AR symptoms last for less than 4 consecutive weeks.
 - Persistent AR is characterized by symptoms that last for more than 4 days a week and for over 4 consecutive weeks.
- **Symptom Severity:**
 - In mild AR, symptoms are non-troublesome, do not impact daily activities, and do not result in sleep disturbance.
 - Severe symptoms can hamper daily activities and work/school performance, potentially leading to absences and decreased efficiency. Severe symptoms are also responsible for further medical complications.

Allergic Rhinitis (AR) Classification:

- **Intermittent Symptoms:**
 - Occurrence: < 4 days a week or for < 4 weeks at a time.
- **Persistent Symptoms:**
 - Occurrence: ≥ 4 days a week and/or for ≥ 4 consecutive weeks.

Grading Based on Symptom Severity:

1. **Mild:**
 - Sleep: Not impacted.
 - Daily Activities: Proceed as usual.
 - Work/School: Regular attendance and performance.
 - No troublesome symptoms experienced.
2. **Moderate-to-Severe:**
 - At least one of the following is present:

- Disturbance in sleep.
- Daily activities are impacted, including a possible restriction in performing them.
- School/work is affected, which might include absence or decreased performance.
- Troublesome symptoms are present.

Pathogenesis:

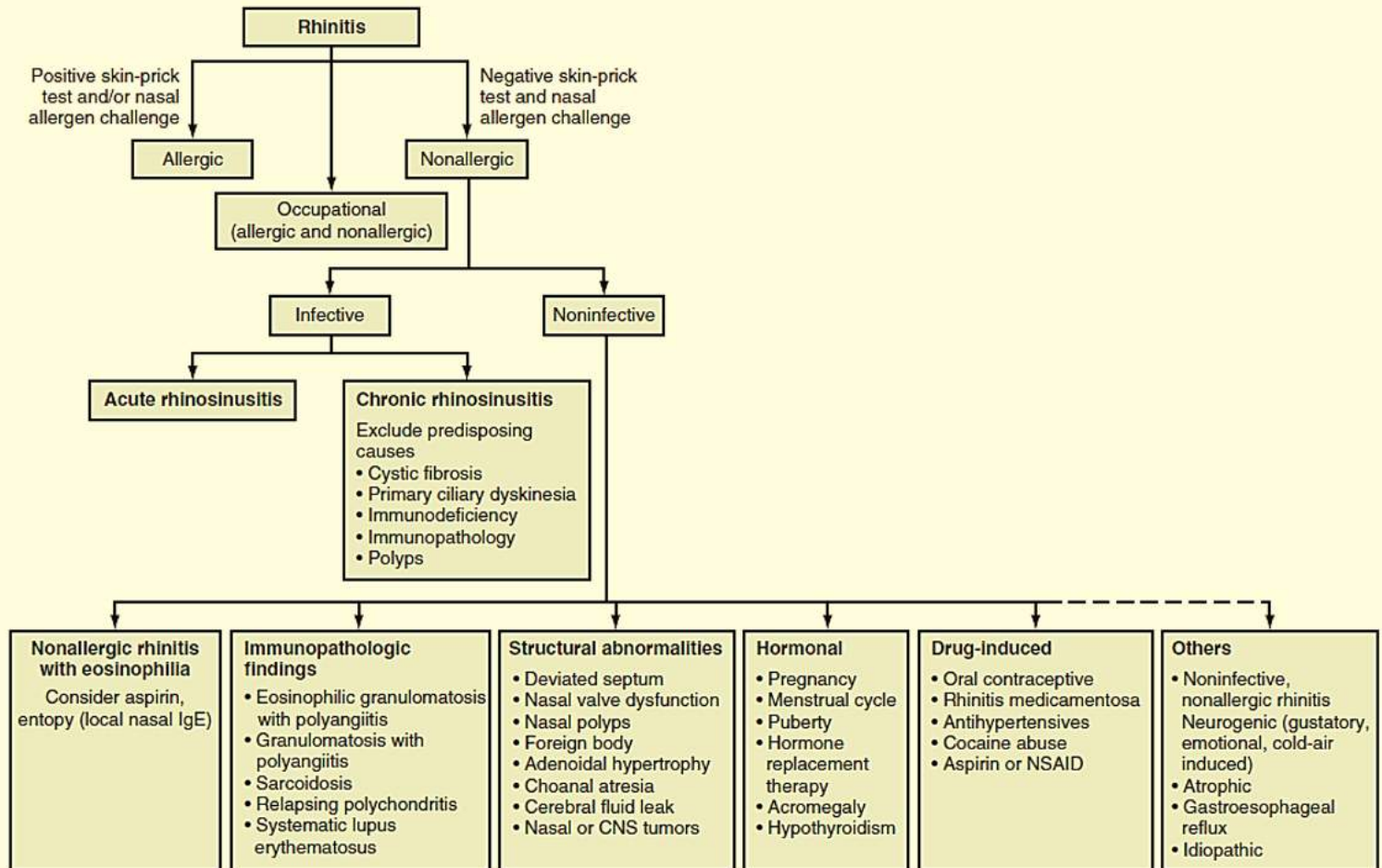
1. Atopic host exposure to an allergen leads to the production of sIgE.
2. sIgE is linked with eczema in childhood and with asthma and rhinitis after age 4.
3. Clinical reactions on allergen reexposure:
 - **Early-phase Allergic Response:**
 - a) Initiated by bridging of the IgE molecules on mast cell surfaces by allergens.
 - b) Characterized by mast cell degranulation and the release of inflammatory mediators such as histamine, prostaglandin 2, and cysteinyl leukotrienes.
 - **Late-phase Allergic Response:**
 - a) Occurs 4-8 hours after allergen exposure.
 - b) Nasal mucosa experiences infiltration by inflammatory cells including basophils, eosinophils, neutrophils, mast cells, and mononuclear cells.
 - c) Eosinophils release proinflammatory mediators and serve as a source of IL-3, IL-5, GM-CSF, and IL-13.
 - d) Allergen reintroduction leads to "priming", causing a stronger response even with lesser provocation.
 - e) A significant increase in submucosal mast cells occurs throughout the allergy season.
 - f) These mast cells, previously thought to only play a role in early-phase response, are essential in chronic allergic disease.

Clinical manifestations:

- Symptoms may be overlooked or mistakenly attributed to respiratory infections.
- **Typical behaviors include:**
 - Nose blowing in older children.
 - Sniffing and snorting in younger children.
 - Nasal itching resulting in grimacing, twitching, and picking of the nose, potentially causing epistaxis.
 - The "allergic salute" involves upward nose rubbing, leading to a transverse nasal crease.
- **Diagnostic criteria:**
 - Symptoms without the presence of an upper respiratory tract infection or structural abnormalities.
- **Typical symptoms include:**
 - Intermittent nasal congestion, itching, sneezing, clear rhinorrhea, and conjunctival irritation.
 - Symptom severity increases with greater allergen exposure.
 - Loss of smell and taste.
 - Potential presence of headaches, wheezing, and coughing.
 - Preschoolers with both chronic wheezing and rhinitis experience more severe wheezing than those without rhinitis.
 - Nasal congestion can worsen at night, causing mouth breathing, snoring, sleep disruption, and irritability.
- **Physical examination findings:**
 - Abnormal facial development, dental malocclusion, continuous open-mouth breathing, chapped lips, dark circles under the eyes, and a transverse nasal crease.
 - Conjunctival edema, itching, tearing, and hyperemia.
 - Nasal examination may reveal clear nasal secretions, edematous membranes, swollen turbinates, and potential blockage of the nasal airway.

Differential diagnosis and approach:

- AR evaluation involves a thorough history, physical examination, and laboratory tests.



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Q: Treatment of allergic rhinitis

Treatment of Allergic Rhinitis

- **Guideline-directed Management**

- ARIA (Allergic Rhinitis and its Impact on Asthma) provides evidence-based treatment.
- Aims to improve disease control.
- Prioritizes quality-of-life measures for symptom evaluation and treatment response assessment.

- **Treatment Goals**

- Provide safe and effective symptom prevention and relief.
- Specific indoor allergen exposure reduction measures can reduce sensitization risks and respiratory disease symptoms.

- **Allergen Exposure Limitations**

- Allergen-proof encasings for mattresses, pillows, and covers can reduce mite allergen exposure.
- Weekly washing of bed linen and blankets in hot water ($>54.4^{\circ}\text{C}$ [130°F]).
- Best measure for animal allergen avoidance is pet removal.
- Pollen and outdoor mold exposure can be minimized by staying indoors in controlled environments.
- Air conditioning keeps doors and windows shut, thus reducing pollen exposure.
- High-efficiency particulate air (HEPA) filters reduce airborne mold spore counts.

- **Medication for Allergic Rhinitis**

- **Oral Antihistamines**

- Used for sneezing, rhinorrhea, and ocular symptom relief.
- Effective for mild-intermittent disease.
- Classified as:
 - First-generation (relatively sedating)

- Second-generation (relatively nonsedating)
- Available for oral, topical ophthalmic, and intranasal administration.
- Both generations available as non-prescription drugs.
- Second-generation antihistamines preferred due to reduced sedation.
- ***Pseudoephedrine***
 - Used for symptom relief, such as nasal congestion, sinus pressure, rhinorrhea, sneezing, lacrimation, itching, and cough.
 - Often found in combination with other agents.
 - Oral vasoconstrictor known to cause irritability and insomnia.
 - Associated with increased infant mortality risks, especially for children 2-3 years old.
 - Misused for methamphetamine and methcathinone synthesis.
- ***Specific Medication for Allergic Rhinitis***
 - ***Anticholinergic Nasal Spray***
 - Ipratropium bromide - used for serous rhinorrhea treatment (see Table 168.5).
 - ***Intranasal Decongestants***
 - Oxymetazoline and phenylephrine - used for less than 5 days and shouldn't be repeated more than once a month to prevent rebound nasal congestion.
 - ***Sodium Cromoglycate***
 - Effective but requires administration every 4 hours.
 - ***Leukotriene-modifying Agents***
 - Modest effect on rhinorrhea and nasal blockage.
 - ***Nasal Saline Irrigation***
 - Beneficial adjunctive treatment for all AR forms.
 - ***Intranasal Corticosteroids***

- Highly effective AR therapy, potentially beneficial for concurrent allergic conjunctivitis.
- Reduces eosinophilic inflammation symptoms in AR but not in rhinitis with neutrophils or without inflammation

Pharmacological Management of Allergic Rhinitis in Pediatric Patients

Drug Class	Drug Name (Generic)	Mechanism of Action	Indications	Dosage and Administration for Pediatrics	Contraindications & Side Effects
First-Generation Antihistamines	Chlorpheniramine	H1 receptor antagonist	Rhinorrhea, sneezing, itching	1 mg every 4-6 hr for children 2-6 years	Sedation, dry mouth
Second-Generation Antihistamines	Cetirizine	H1 receptor antagonist	Rhinorrhea, sneezing, itching	2.5-5 mg/day for children 2-6 years	Less sedating, caution in renal impairment
	Levocetirizine	H1 receptor antagonist	Rhinorrhea, sneezing, itching	1.25 mg/day for children 6 months-5 years	Less sedating, caution in renal impairment
	Desloratadine	H1 receptor antagonist	Rhinorrhea, sneezing, itching	1.25 mg/day for children 1-5 years	Less sedating, caution in renal impairment
Leukotriene Antagonist	Montelukast	Blocks leukotriene receptors	Rhinorrhea, nasal blockage	4 mg/day for children 2-5 years	Neuropsychiatric events, liver enzyme elevation
Intranasal Corticosteroids	Fluticasone	Anti-inflammatory	Rhinorrhea, sneezing, itching	1 spray/nostril/day for children 4-11 years	Epistaxis, nasal dryness
	Mometasone	Anti-inflammatory	Rhinorrhea, sneezing, itching	1 spray/nostril/day for children 2-11 years	Epistaxis, nasal dryness

	Beclomethasone	Anti-inflammatory	Rhinorrhea, sneezing, itching	1 spray/nostril/day for children 6-12 years	Epistaxis, nasal dryness
Decongestants	Pseudoephedrine	Alpha-adrenergic agonist	Nasal congestion	15-30 mg every 4-6 hr for children 2-6 years	Hypertension, insomnia
	Oxymetazoline	Alpha-adrenergic agonist	Nasal congestion	2-3 sprays/nostril every 12 hr for children 6-12 years	Rebound congestion if used >5 days
Anticholinergics	Ipratropium Bromide	Inhibits acetylcholine receptors	Rhinorrhea	1 spray/nostril 2-3 times/day for children 6 years and older	Dry mouth, nasal dryness
Mast Cell Stabilizers	Cromolyn Sodium	Inhibits mast cell degranulation	Prophylaxis	1 spray/nostril 4-6 times/day for children 2 years and older	Minimal side effects, frequent dosing

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Q: Use of Nebulizers in Pediatric practice and Aerosol therapy in children

Criteria	Nebulizer	Aerosol Therapy
Device Type	Electrical or battery-operated device	Metered-Dose Inhalers (MDIs), Dry Powder Inhalers (DPIs)
Mechanism of Action	Atomizes liquid medication into mist	Propellant-driven or breath-actuated release
Medication Form	Liquid solutions or suspensions	Pressurized liquid or dry powder

Indications	Acute conditions, younger children, severe asthma	Maintenance treatment, older children, mild to moderate asthma
Dosage Control	Flexible, allows for titration	Fixed-dose per actuation
Administration Time	5-10 minutes	Less than a minute
Technique Sensitivity	Less dependent on technique	Requires coordinated actuation and inhalation
Portability	Less portable	Highly portable
Cost	Higher initial cost, cheaper medication	Lower device cost, more expensive medication
Maintenance	Regular cleaning required	Minimal, may require spacers
Side Effects	Potential for more systemic absorption, risk of bacterial contamination	Lower systemic absorption, risk of oral thrush
Patient Age Suitability	Suitable for all ages, including infants	Best for children who can follow instructions
Efficacy Monitoring	Periodic assessment required	Built-in dose counters in some devices
Environmental Impact	Lower, no propellants	Some MDIs use propellants
Compliance and Adherence	Longer administration may affect adherence	Easier and quicker, potentially better adherence
Drug Availability	Broader range of medications	Limited to formulations compatible with device
Particle Size	Variable, can be adjusted	Fixed, determined by device
Drug Waste	Potential for more waste	More efficient delivery
Rescue vs. Maintenance	Often used for rescue medication	Primarily used for maintenance therapy
Healthcare Supervision	May require more frequent monitoring	Less frequent monitoring needed

Ease of Use	May require assembly	Pre-assembled
Drug-Drug Interactions	Potential for interactions due to higher systemic absorption	Lower risk due to lower systemic absorption
Patient Training Required	Yes, on assembly and cleaning	Yes, on actuation and inhalation techniques

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Q: Define bronchial asthma classify bronchial asthma in children

Definition of Bronchial Asthma

- **Bronchial Asthma:** A chronic inflammatory disorder of the airways characterized by recurrent episodes of wheezing, breathlessness, chest tightness, and coughing. The condition involves a complex interplay of airway hyperresponsiveness, inflammation, and airflow obstruction.

Classification:

Classification Basis	Sub-Type	Characteristics
Severity	Intermittent Asthma	- Symptoms less than once a week - Brief episodes - No symptoms between episodes
	Mild Persistent Asthma	- Symptoms more than once a week but less than once a day - Episodes may affect activity
	Moderate Persistent Asthma	- Daily symptoms - Episodes affect activity - Exacerbations may require emergency intervention
	Severe Persistent Asthma	- Continual symptoms - Frequent exacerbations - Frequent nocturnal symptoms
Phenotype	Allergic Asthma	- Triggered by allergens like pollen, dust mites - Often associated with hay fever
	Non-Allergic Asthma	- Triggered by factors like infections, exercise, cold air

		- No underlying allergies
	Exercise-Induced Asthma	- Triggered by physical exertion or exercise
	Cough-Variant Asthma	- Chronic cough as the sole symptom
	Transient Nonatopic Wheezing	- Common in early preschool years - Triggered by respiratory viral infections - Resolves by school age
	Persistent Atopy-Associated Asthma	- Begins in early preschool years - Associated with atopy - High risk for persistence
	Asthma with Declining Lung Function	- Progressive increase in airflow limitation - Associated with hyperinflation, male gender
Age of Onset	Early-Onset Asthma	- Symptoms appear before age 5 - Often associated with eczema or allergic rhinitis
	Late-Onset Asthma	- Symptoms appear after age 5 - May be triggered by respiratory infections or environmental factors
Control	Well-Controlled Asthma	- Symptoms are rare and mild - No limitations on activities
	Partially Controlled Asthma	- Some limitations on activities - Symptoms may require use of rescue medication
	Uncontrolled Asthma	- Frequent severe episodes - Significant limitations on activities
Response to Treatment	Steroid-Responsive Asthma	- Symptoms improve with corticosteroid treatment
	Steroid-Resistant Asthma	- Symptoms do not improve significantly with corticosteroid treatment
Management Patterns	Easy-to-Control	- Well controlled with low levels of daily controller therapy
	Difficult-to-Control	- Well controlled with multiple and/or high levels of controller therapies
	Exacerbators	- Despite being well controlled, continue to have severe exacerbations

	Refractory	- Continue to have poorly controlled asthma despite multiple and high levels of controller therapies
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Q: Risk Factors and pathology of Asthma

Risk Factors for persistence of Asthma

- Parental asthma
- Allergy:
 - Atopic dermatitis (eczema)
 - Allergic rhinitis
 - Food allergy
 - Inhalant allergen sensitization
 - Food allergen sensitization
- Severe lower respiratory tract infection:**
 - Pneumonia
 - Bronchiolitis requiring hospitalization
- Wheezing apart from colds
- Male gender
- Low birthweight
- Environmental tobacco smoke exposure
- Reduced lung function at birth
- Formula feeding rather than breastfeeding

Pathophysiology:

Pathophysiology of bronchial asthma

1. **Inflammatory Cascade Initiation:** Exposure to triggers such as allergens, irritants, or infections activates innate and adaptive immune responses. This involves dendritic cells, T-helper cells, and antigen-presenting cells.
2. **Cytokine and Chemokine Release:** Activation of immune cells leads to the secretion of various cytokines (e.g., IL-4, IL-5, IL-13) and chemokines. These molecules act as signaling agents, recruiting more inflammatory cells to the site of inflammation.

3. **Eosinophilic Infiltration:** One of the key features is the recruitment of eosinophils into the airway tissues, facilitated by cytokines like IL-5. Eosinophils release toxic granules that damage the airway epithelium.
4. **Mast Cell Activation:** Mast cells release mediators like histamine, leukotrienes, and prostaglandins, which contribute to bronchoconstriction, increased vascular permeability, and mucus secretion.
5. **Airway Hyperresponsiveness:** The airways become overly sensitive to various stimuli, including cold air, exercise, and even emotional stress. This hyperresponsiveness is partly due to the sensitization of nerve endings in the airways.
6. **Bronchoconstriction:** Smooth muscle cells surrounding the airways contract excessively, narrowing the airway lumen. This is often reversible with bronchodilator treatment but can become persistent in chronic cases.
7. **Mucus Hypersecretion:** Goblet cells in the airway epithelium produce excessive amounts of mucus, contributing to airway obstruction. This mucus can also serve as a medium for bacterial growth, leading to infections.
8. **Airway Remodeling:** Chronic inflammation leads to structural changes in the airway, including fibrosis, smooth muscle hypertrophy, and angiogenesis. These changes can make asthma more resistant to treatment and are generally irreversible.
9. **Gas Exchange Impairment:** The combined effects of bronchoconstriction, mucus plugging, and alveolar collapse can lead to impaired gas exchange, manifesting as decreased oxygen and increased carbon dioxide levels in the blood.
10. **Systemic Effects:** Chronic asthma can have systemic implications, including fatigue, weight loss, and in extreme cases, "air trapping" leading to pneumothorax or respiratory failure.
11. **Genetic and Epigenetic Factors:** Polymorphisms in genes related to inflammation, immune response, and bronchial structure can predispose individuals to asthma. Epigenetic changes due to environmental exposures can also modulate asthma risk and severity.
12. **Neuroendocrine Regulation:** Stress and hormones can modulate the severity and frequency of asthma attacks. The hypothalamic-pituitary-adrenal axis and sympathetic nervous system have roles in this aspect.

13. **Co-morbid Conditions:** Asthma often coexists with other conditions like allergic rhinitis, obesity, and GERD, which can complicate its pathophysiology and management.
14. **Pharmacological Implications:** Understanding the complex pathophysiology of asthma is crucial for the development of targeted therapies, including monoclonal antibodies against specific cytokines and small-molecule inhibitors against various signaling pathways.
15. **Diagnostic Challenges:** The heterogeneous nature of asthma, with various phenotypes and endotypes, makes diagnosis and treatment a clinical challenge. Biomarkers like FeNO, sputum eosinophils, and serum IgE levels are used for more precise characterization.

Pathogenesis:

1. **Epithelial Cell Activation:**

- The process begins with the activation of epithelial cells due to the presence of allergens and viruses.
- This activation leads to the production of proinflammatory cytokines and chemokines.

2. **TSLP (Thymic Stromal Lymphopoietin) Production:**

- Activated epithelial cells release TSLP, which in turn stimulates the production of IL-9, a cytokine associated with mucous production.

3. **Dendritic Cells (DC):**

- DCs in the submucosa play a pivotal role in sensing allergens and triggering immune responses.
- This diagram shows their interaction with T helper 2 (Th2) cells via OX40L, which results in the release of various cytokines.

• **Role of Chemokines:**

- Chemokines play a key role in the adhesion and tissue migration of Th2 cells and eosinophils. They facilitate the movement of these immune cells to the site of inflammation.

4. **Th2 Cells:**

- Th2 cells are central to the asthmatic response. Once activated, they produce a range of cytokines including IL-4, IL-5, IL-9, IL-13, and others.

- IL-4 and IL-13, in particular, stimulate B cells to produce IgE, which plays a role in allergic reactions.

5. ***Mast Cells and Basophils:***

- Both cell types possess IgE receptors on their surface.
- When IgE binds to these receptors, it triggers the release of inflammatory mediators that contribute to bronchial hyperreactivity and other asthmatic symptoms.

6. ***Role of Eosinophils:***

- Elevated in many asthmatic individuals, eosinophils play a role in tissue inflammation. The cytokine IL-5, produced by Th2 cells, regulates tissue eosinophilia.

7. ***Other Immune Cells:***

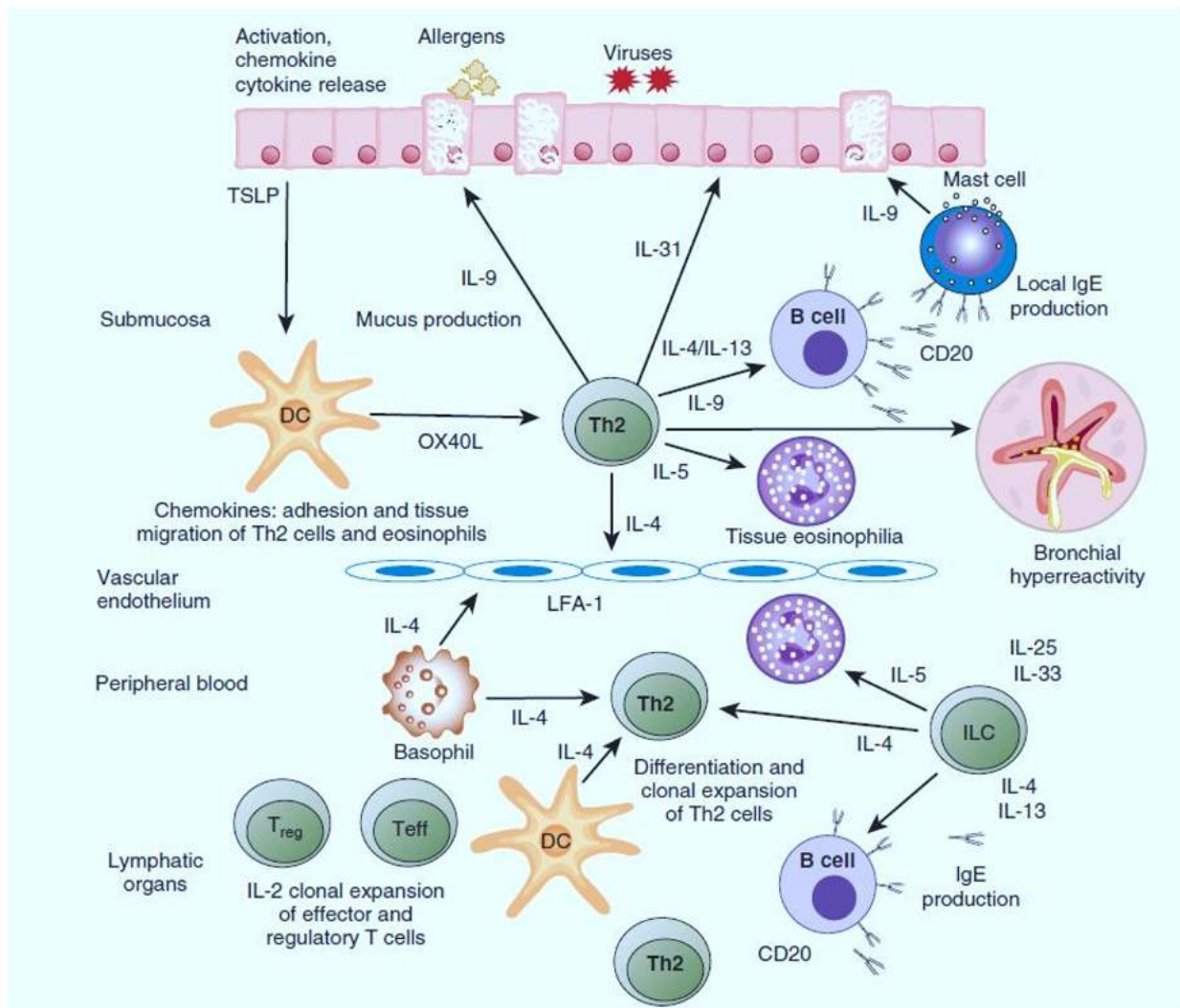
- Roles of regulatory T cells (Treg), effector T cells (Teff), and innate lymphoid cells (ILC).
- These cells interact in a complex manner to regulate immune responses in asthma.

8. ***Vascular and Peripheral Blood Interactions:***

- Role of LFA-1 in immune cell migration and adhesion.

9. ***Bronchial Hyperreactivity:***

- Central to asthma, bronchial hyperreactivity is depicted as a consequence of the cascading immune reactions initiated by allergens and viruses.



Q: Approach to a child with recurrent wheezing.

- ❖ The approach to a child with recurrent wheezing involves a comprehensive assessment to determine the underlying cause and appropriate management strategies.
- ❖ Recurrent wheezing is common in children and can be due to various reasons, including asthma, viral infections, anatomical anomalies, and other medical conditions.
- ❖ A systematic approach, including a detailed history, physical examination, and appropriate investigations, is essential for accurate diagnosis and management.

1. **Detailed Medical History:**

- a) Frequency, duration, and severity of wheezing episodes.
- b) Associated symptoms (e.g., cough, breathlessness, chest tightness).
- c) Triggers (e.g., exercise, allergens, cold air, infections).
- d) Family history of asthma or atopic diseases.
- e) Response to previous treatments.

2. **Physical Examination:**

- a) Assess respiratory rate, oxygen saturation, use of accessory muscles.
- b) Listen for wheezing, crackles, or decreased air entry.
- c) Look for signs of atopy (e.g., eczema, allergic rhinitis).
- d) Evaluate for growth and developmental delays.

3. **Diagnostic Investigations:**

- a) **Pulmonary Function Tests (PFTs):** Spirometry in children old enough to perform the test (usually >5 years old).
- b) **Chest X-Ray:** To rule out structural abnormalities and other lung conditions.
- c) **Allergy Testing:** Skin prick tests or serum IgE levels to identify potential allergens.
- d) **Blood Tests:** Complete blood count (CBC) to check for eosinophilia; other tests based on clinical suspicion.

4. **Identifying Underlying Causes:**

- a) Asthma: The most common cause of recurrent wheezing.
- b) Viral-induced wheeze: Common in preschool children.
- c) Anatomical anomalies: Such as vascular rings, tracheomalacia.
- d) Other causes: Cystic fibrosis, foreign body aspiration, immune deficiencies.

5. **Management Strategies:**

- a) **Asthma:** Follow asthma guidelines for management, including the use of inhaled corticosteroids, bronchodilators, and leukotriene receptor antagonists.

- b) **Viral-induced wheeze:** Symptomatic treatment; bronchodilators may be helpful in some cases.
- c) **Allergy Management:** Avoidance of identified allergens, use of antihistamines or nasal corticosteroids for allergic rhinitis.
- d) **Education and Monitoring:** Educate the family about the condition, trigger avoidance, and the correct use of inhalers. Provide an action plan for exacerbations.

6. **Regular Follow-Up:**

- a) Monitor response to treatment, growth, and development.
- b) Adjust treatment based on control of symptoms and side effects.

7. **Referral to Specialists:**

- a) Consider referral to a pediatric pulmonologist or allergist for complex cases or if the diagnosis is uncertain.
- b) Referral for further diagnostic evaluation in cases of suspected anatomical anomalies or other rare causes.

8. **Preventive Measures:**

- a) Vaccinations: Ensure up-to-date vaccinations, including the influenza vaccine.
- b) Exposure to tobacco smoke: Advise against smoking in the child's environment.

For addressing the differential diagnoses:

Condition	Key Features	Distinguishing Factors
Bronchial Asthma	Episodic wheezing, cough, dyspnea. Triggered by allergens, exercise, cold air.	Family history of atopy. Good response to bronchodilators.
Viral Bronchiolitis	Common in infants and young children. Associated with viral upper respiratory infections.	Seasonal occurrence, often in winter. Rapid onset with respiratory distress.
Foreign Body Aspiration	Sudden onset of wheezing in a previously healthy child. May have a history of choking.	Unilateral wheezing or decreased breath sounds. No response to asthma medications.

Gastroesophageal Reflux Disease (GERD)	Wheezing, cough, especially postprandial or nocturnal. May have symptoms of acid reflux.	Symptoms improve with anti-reflux treatment. May coexist with asthma.
Tracheomalacia/Bronchomalacia	Congenital weakness of the trachea/bronchial walls. Wheezing, stridor, barking cough.	Symptoms present from early infancy. May improve with age.
Cystic Fibrosis	Persistent wheezing, cough with sputum. Failure to thrive, recurrent infections.	Positive family history. Positive sweat chloride test.
Vocal Cord Dysfunction	Paradoxical vocal cord movement. Stridor, throat tightness, voice changes.	Symptoms can mimic asthma but do not respond to bronchodilators. Diagnosed by laryngoscopy.
Allergic Rhinitis/Sinusitis	Nasal symptoms, postnasal drip, cough. Seasonal or perennial allergies.	Associated with other allergic symptoms. Response to antihistamines or nasal corticosteroids.
Cardiac Asthma	Wheezing due to heart failure. Associated with exercise intolerance, fatigue.	Cardiac history or findings on examination. Abnormal cardiac investigations.
Bronchopulmonary Dysplasia	In preterm infants with a history of prolonged mechanical ventilation. Chronic respiratory distress, wheezing.	History of prematurity and neonatal intensive care unit stay. Abnormal chest X-ray findings.
Immunodeficiency Disorders	Recurrent infections, wheezing, failure to thrive.	History of severe, recurrent, or unusual infections. Laboratory evidence of immunodeficiency.

Notes:

- **History and Physical Examination:** A thorough history and physical examination are crucial in differentiating these conditions.
- **Diagnostic Testing:** May include chest X-ray, pulmonary function tests, allergy testing, sweat chloride test, or bronchoscopy, depending on the suspected diagnosis.

- **Multidisciplinary Approach:** In complex cases, collaboration with specialists such as pulmonologists, allergists, gastroenterologists, or cardiologists may be necessary.

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Q: Clinical Features of Bronchial Asthma in Children

1. **Recurrent Wheezing:** Audible high-pitched whistling sound during breathing, often exacerbated by viral infections or exercise.
2. **Cough:** Persistent, often worse at night, may be the only symptom in some children.
3. **Shortness of Breath:** Difficulty in breathing, often noticed during physical activities or at night.
4. **Chest Tightness:** Subjective feeling of constriction in the chest area.
5. **Tachypnea:** Increased rate of breathing, often accompanied by nasal flaring and use of accessory muscles.
6. **Reduced Exercise Tolerance:** Children may avoid physical activities due to breathlessness.
7. **Nocturnal Symptoms:** Worsening of symptoms like cough and wheezing during the night, affecting sleep quality.
8. **Seasonal Variations:** Symptoms may worsen during specific seasons due to allergen exposure.
9. **Family History:** Often a history of atopy or asthma in the family.
10. **Exacerbation Triggers:** Identification of specific triggers like cold air, smoke, or allergens.
11. **Response to Bronchodilators:** Rapid relief of symptoms with the use of inhaled bronchodilators can be indicative.

The following table encapsulates the various criteria we commonly use for assessing the severity of bronchial asthma symptoms and functional assessment, ranging from mild to severe, including a subset indicating imminent respiratory arrest.

Criteria/Severity Level	Mild	Moderate	Severe	Subset (Respiratory Arrest Imminent)
Breathlessness	Occurs while walking	Occurs at rest, observed in infants as softer, shorter cry, difficulty feeding	Occurs at rest, where infants stop feeding	Extreme dyspnea and anxiety
Talks in...	Sentences	Phrases	Words	Upright, leaning forward, unable to talk
Signs of Respiratory Distress	May be agitated	Usually agitated	Extremely agitated	Drowsy or confused
Accessory Muscle Use/Suprasternal Retractions	Absent	Increased	Commonly	Often >30 breaths/min
Wheeze	Moderate; often only end-expiratory	Loud	Usually loud; throughout inhalation and exhalation	Paradoxical thoracoabdominal movement
Pulse Rate (beats/min) & Pulsus Paradoxus	<100 beats/min, pulsus paradoxus absent	100-120 beats/min, may be present	>120 beats/min, often present	Bradycardia (in adults), absence suggests respiratory muscle fatigue
Expiratory Flow Value (PEF, % predicted or personal best)	≥70%	Approximately 40-69% of response, might last >2 hours	<60% with possible cyanosis	<25%
PaO₂ & SaO₂ at Sea Level	Normally not tested	≥42 mm Hg (test not usually necessary), 90-95%	<42 mm Hg, <90%	Hypoxia despite oxygen therapy

Differential Diagnosis of Bronchial Asthma in Children

- **Bronchiolitis:** Common in infants, usually caused by viral infections like RSV, characterized by wheezing and respiratory distress.

- **Tuberculosis:** Chronic cough, night sweats, and weight loss, more common in endemic areas. It has to be one of the top differentials when we are dealing with patients in India because once steroids are started it will mask the symptoms and worsen the disease progression if there is an underlying tuberculosis.
- **Foreign Body Aspiration:** Sudden onset of coughing, choking, and unilateral wheezing.
- **Vocal Cord Dysfunction:** Paradoxical vocal cord movement leading to stridor, often mistaken for asthma.
- **Gastroesophageal Reflux Disease (GERD):** Chronic cough and wheezing, symptoms may improve with antacid treatment.
- **Cystic Fibrosis:** Persistent cough, failure to thrive, and recurrent respiratory infections.
- **Congenital Heart Disease:** Symptoms like tachypnea and wheezing, but often accompanied by other signs like cyanosis.
- **Tracheomalacia and Bronchomalacia:** Congenital conditions leading to dynamic airway collapse, symptoms may mimic asthma.
- **Chronic Sinusitis:** May present with chronic cough and wheezing, often associated with nasal symptoms.
- **Pulmonary Hypertension:** Rare in children, but can present with shortness of breath and exercise intolerance.
- **Allergic Rhinitis:** May coexist with asthma, but isolated symptoms like sneezing and nasal congestion are more prominent.
- **Psychogenic Cough:** Chronic cough without other respiratory symptoms, often exacerbated by stress or anxiety.
- **Pneumonia:** Acute onset with fever, localized crackles on auscultation, confirmed by chest X-ray.

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Q: Lab evaluation for asthma

Test/Parameter	Description	Diagnostic Criteria	Additional Notes
Pulmonary Function Testing			
Forced Expiratory Airflow Measures	Helpful in diagnosing and monitoring asthma	Particularly useful in children who are poor perceivers of airflow obstruction	
Spirometry	Measures airflow and lung volumes during forced expiratory maneuvers	Essential in children at risk for severe asthma exacerbations and those with poor perception of symptoms	Feasible in children >6 years old
FEV1/FVC Ratio	Ratio of forced expiratory volume in 1 sec to forced vital capacity	<0.80 indicates airflow obstruction	Normative values determined by height, gender, and ethnicity
Bronchodilator Response			
Improvement in FEV1	After inhalation of a short-acting β -agonist (SABA)	$\geq 12\%$ improvement is consistent with asthma	
Bronchoprovocation Challenges			
Airway Hyperresponsiveness (AHR)	Sensitivity to inhaled methacholine, mannitol, and cold or dry air	Degree of AHR correlates with asthma severity and airways inflammation	Rarely practical in general practice
Exercise Challenges	Identify children with exercise-induced bronchospasm	FEV1 decreases during or after exercise by >15%	Onset usually within 5 min, peaks at 15 min, resolves within 30-60 min
Peak Expiratory Flow (PEF) Monitoring			
PEF Devices	Measure airflow at home	Diurnal variation in PEF >20% is	Less sensitive and reliable than spirometry

		consistent with asthma	
Exhaled Nitric Oxide (FeNO)			
FeNO Levels	Noninvasive measure of allergic airways inflammation	>20 ppb supports clinical diagnosis of asthma	Can predict response to inhaled corticosteroid (ICS) therapy
Radiology			
Chest Radiographs	Identify abnormalities and complications	Often appear normal, except for subtle signs of hyperinflation and peribronchial thickening	Can help identify asthma mimics and complications; TB possibility
High-Resolution CT Scans			Can better appreciate lung abnormalities like bronchiectasis; Rules out TB
Allergy Testing			
Skin-Prick Testing	Assess sensitization to inhalant allergens	88% of asthmatic children aged 5-12 had inhalant allergen sensitization	Part of the Childhood Asthma Management Program (CAMP) study

For addressing some of the differential diagnosis:

Test/Parameter	Purpose	Diagnostic Criteria/Findings	Differential Diagnosis Ruled Out	Additional Notes
Pulmonary Function Tests (PFTs)	Evaluate lung function	FEV1/FVC ratio <0.80 indicates airflow obstruction	COPD, Bronchiectasis	Spirometry is the most common PFT

Bronchodilator Reversibility Test	Assess reversibility of airflow obstruction	Improvement in FEV1 $\geq$ 12% after bronchodilator use	Fixed airway obstruction	Often done as part of spirometry
Peak Expiratory Flow (PEF) Monitoring	Assess airflow at home	Diurnal variation in PEF >20% is consistent with asthma	Not specific; cannot rule out other conditions	Less sensitive than spirometry
Exhaled Nitric Oxide (FeNO)	Measure allergic airways inflammation	>20 ppb supports asthma diagnosis	GERD, VCD, Cystic Fibrosis	Noninvasive; can be done in children as young as 5
Bronchoprovocation Tests	Evaluate airway hyperresponsiveness	Positive to methacholine, mannitol, or exercise	Healthy individuals or other lung diseases	Rarely used in general practice
Chest X-ray	Identify structural abnormalities	Hyperinflation, peribronchial thickening	Pneumonia, TB, pneumothorax, heart failure	Often appears normal in asthma
High-Resolution CT Scan	Detailed lung imaging	Bronchiectasis, air trapping	Cystic fibrosis, allergic bronchopulmonary aspergillosis	More sensitive than X-ray
Allergy Testing (Skin or Blood)	Identify allergic triggers	Positive to specific allergens	Non-allergic asthma	Can guide environmental control measures
Complete Blood Count (CBC)	Assess for infection or inflammation	Elevated eosinophils may support asthma diagnosis	Infections, other inflammatory conditions	Not specific for asthma
Sputum Culture and Sensitivity	Identify bacterial infection	No bacterial growth supports asthma	Bacterial pneumonia, bronchitis	Not routinely done

Echocardiogram	Evaluate heart function	Normal in asthma	Heart failure, pulmonary hypertension	To rule out cardiac causes of symptoms
Arterial Blood Gas (ABG)	Assess severity of respiratory distress	Respiratory acidosis in severe exacerbations	COPD, metabolic disorders	Usually done in emergency settings

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Q: Pulmonary Function Tests (PFTs) in Childhood Asthma

- PFTs are integral in diagnosing asthma, especially in atypical or unclear cases.
- Regular PFTs can help in identifying poorly controlled asthma and the need for therapy modification.
- Patient education on technique, especially for home monitoring tools like peak flow meters, is crucial for accurate assessment.
- PFTs should be interpreted in the context of clinical history, symptoms, and response to asthma medications.

Purpose: Assess lung function, diagnose asthma, monitor response to therapy.

Types: Spirometry, Peak Expiratory Flow Rate (PEFR), Bronchoprovocation tests.

Spirometry

- **Parameters Measured:**
 - Forced Vital Capacity (FVC): Total volume of air exhaled forcefully.
 - Forced Expiratory Volume in 1 second (FEV1): Volume exhaled in the first second of FVC.
 - FEV1/FVC Ratio: Percentage of lung capacity exhaled in one second.
- **Interpretation:**
 - **Obstructive Pattern:** Reduced FEV1/FVC ratio (<75-80% in children).
 - **Reversibility Test:** Increase in FEV1 by ≥12% and 200 mL post-bronchodilator indicates asthma.
- **Age Consideration:** Reliable in children aged ≥5 years.

Peak Expiratory Flow Rate (PEFR)

- **Measurement:** Using a peak flow meter, measures the maximum speed of expiration.
- **Usage:** Home monitoring, assess severity, and response to treatment.
- **Interpretation:**
 - Personal best PEF established during periods of good asthma control.
 - Reduction from personal best indicates worsening control.

Bronchoprovocation Tests

- **Types:**
 - Methacholine Challenge: Inhalation of methacholine to induce bronchoconstriction.
 - Exercise Challenge: Identifies exercise-induced bronchoconstriction.
- **Indication:** When spirometry is inconclusive but asthma is suspected.
- **Interpretation:** Decrease in FEV1 by 10-20% indicates hyperresponsiveness.

Fractional Exhaled Nitric Oxide (FeNO)

- **Measurement:** Non-invasive marker of eosinophilic airway inflammation.
- **Indication:** Assessing inflammation, predicting steroid response.
- **Interpretation:** Elevated levels suggest eosinophilic inflammation.

Impulse Oscillometry (IOS)

- **Application:** Measures lung function during normal breathing, useful in younger children.
- **Parameters:** Resistance and reactance at various frequencies.
- **Advantage:** Easier for young children who cannot perform spirometry.

Age-Specific Considerations

- **Young Children (<5 years):** Limited ability to perform spirometry, rely on clinical diagnosis and response to therapy.
- **School-Aged Children:** Spirometry is the gold standard.

Monitoring and Follow-Up

- **Regular Assessment:** Monitor lung function to guide therapy adjustments.
- **Post-Treatment Evaluation:** Assess response to asthma medications.

- **Long-Term Monitoring:** Identify trends in lung function, assess control.

Challenges and Limitations

- **Technique Dependent:** Requires proper technique and patient cooperation.
- **Interpretation Variability:** Results can vary based on effort, technique, and equipment.
- **Limited in Young Children:** Under 5 years, spirometry is often not feasible.

Role in Asthma Management

- **Diagnosis:** Confirms obstructive lung disease and reversibility.
- **Treatment Decisions:** Guides step-up or step-down of therapy.
- **Education and Self-Management:** Helps in understanding asthma control.

Q: Treatment of Bronchial Asthma in Children

Component 1: Regular Assessment and Monitoring

- **Spirometry:** Conducted at diagnosis, after treatment to assess control, and periodically to monitor lung function.
- **Peak Flow Monitoring:** Useful for daily self-assessment at home.
- **Symptom Monitoring:** Use of validated questionnaires like the Asthma Control Test (ACT).
- **Exhaled Nitric Oxide:** Measures airway inflammation, useful in treatment adjustments.

Component 2: Patient Education

- **Asthma Action Plans:** Individualized plans detailing medication and action in different scenarios.
- **Inhaler Technique:** Regular training on proper inhaler technique.
- **Disease Awareness:** Education on asthma triggers, symptoms, and the importance of adherence.

Adherence

- **Regular Follow-ups:** To assess medication adherence.

- **Educational Interventions:** To improve adherence rates.
- **Electronic Monitoring Devices:** To track inhaler usage.

Component 3: Control of Factors Contributing to Asthma Severity

Eliminating and Reducing Problematic Environmental Exposures

- **Allergen Avoidance:** Dust mites, pet dander, and pollen.
- **Smoke Exposure:** Strong recommendation for smoke-free environments.

Treating Comorbid Conditions

- **Allergic Rhinitis:** Treated with intranasal corticosteroids.
- **GERD:** Managed with antacids and lifestyle modifications.
- **Obesity:** Weight management programs.

Component 4: Principles of Asthma Pharmacotherapy

"Step-Up, Step-Down" Approach

- **Step-Up:** Escalation of treatment if control is not achieved.
- **Step-Down:** De-escalation once control is maintained.

Referral to Asthma Specialist

- **Severe Cases:** Referral for specialized care.
- **Uncontrolled Symptoms:** Despite standard treatment.

Long-Term Controller Medications:

Inhaled Corticosteroids

- **First-Line:** For persistent asthma.
- **Dosing:** Varies based on age and severity.

Systemic Corticosteroids

- **Short Courses:** For acute exacerbations.
- **Monitoring:** For side effects like growth suppression.

Long-Acting Inhaled β -Agonists

- **Not as Monotherapy:** Always in combination with ICS.

Combination ICS/LABA Therapy

- **Advantage:** Improved compliance and possibly better control.

Leukotriene-Modifying Agents

- **Alternative:** To low-dose ICS.

Nonsteroidal Antiinflammatory Agents

- **Cromolyn Sodium:** Rarely used due to limited efficacy.

Long-Acting Inhaled Anticholinergics

- **Tiotropium:** For ages 12 and up as add-on therapy.

Allergen Immunotherapy

- **Subcutaneous:** For allergic asthma with a known trigger.

Biologic Therapies:

- **Omalizumab:** Anti-IgE, for severe allergic asthma.
- **Mepolizumab:** Anti-IL-5, for eosinophilic asthma.
- **Dupilumab:** Anti-IL-4, for moderate-to-severe asthma.

Quick-Reliever Medications:

Short-Acting Inhaled β -Agonists

- **Albuterol:** Drug of choice for acute relief.

Anticholinergic Agents

- **Ipratropium Bromide:** For acute severe asthma, usually in combination with SABA.

Delivery Devices and Inhalation Technique

- **Metered-Dose Inhalers:** Most common.
- **Dry Powder Inhalers:** Alternative to MDIs.
- **Nebulizers:** For those unable to use inhalers.

Asthma Exacerbations and Their Management:

Home Management of Asthma Exacerbations

- **SABA:** Immediate use.
- **Contact Healthcare Provider:** If no improvement.

Emergency Department Management of Asthma Exacerbations

- **Oxygen Therapy:** To maintain SpO₂ > 90%.

- **Systemic Corticosteroids:** Early administration.

Hospital Management of Asthma Exacerbations

- **Intensive Monitoring:** Including arterial blood gases.
- **Intravenous Corticosteroids:** In severe cases.

Special Management Circumstances during Surgery

- **Preoperative Assessment:** To assess asthma control.
- **Perioperative Medications:** Including bronchodilators and corticosteroids.
- **Anesthesia Considerations:** Use of inhaled anesthetics that are bronchodilators.

NIH Guidelines for Pediatric Asthma Management

- **Step 1:** Intermittent asthma is managed with short-acting beta-2 agonists like Albuterol and Levalbuterol. These bronchodilators have a quick onset of action within 5-15 minutes and last for 4-6 hours.
- **Step 2:** For persistent asthma, low-dose inhaled corticosteroids (ICS) are preferred. Montelukast is an alternative.
- **Step 3:** Medium-dose ICS is preferred in children aged 0-4 years. For children aged 5-11, either medium-dose ICS or a combination of ICS + long-acting beta-agonist (LABA) or leukotriene receptor antagonist (LTRA) is recommended.
- **Step 4:** Medium-dose ICS + either LABA or Montelukast is recommended for children aged 0-4. For children aged 5-11 and above, medium-dose ICS + LABA is preferred.
- **Step 5:** High-dose ICS + either LABA or Montelukast for ages 0-4; high-dose ICS + LABA for ages 5-11 and above. Omalizumab may also be considered for children aged 12 and above.
- **Step 6:** High-dose ICS + either LABA or Montelukast or oral corticosteroids for ages 0-4; high-dose ICS + LABA + oral systemic corticosteroid for ages 5-11; high-dose ICS + LABA + oral corticosteroid for ages 12 and up.
- **Acute Exacerbations:** Initial management includes bronchodilators and steroids. Nebulized Albuterol doses range from 2.5-5 mg and can be re-dosed every 20 minutes. Ipratropium dosing of 250 to 500 mcg should be co-administered with Albuterol for three doses in moderate to severe exacerbations.

- **Asthma Action Plans:** Recommended for all patients, these are individualized plans developed in partnership with each patient, detailing how to manage asthma under various conditions.
- **Alternative Medications:** Theophylline may be an alternative in Steps 2 through 6 but requires caution due to its narrow therapeutic range.

GINA Guidelines for Pediatric Asthma Management

- **Step 1:** Short-acting beta-2 agonists (SABA) as needed.
- **Step 2:** Regular low-dose inhaled corticosteroids (ICS) or leukotriene receptor antagonists (LTRA) like Montelukast.
- **Step 3:** Low-dose ICS plus long-acting beta-2 agonist (LABA) or medium-dose ICS.
- **Step 4:** Medium-dose ICS plus LABA.
- **Step 5:** High-dose ICS plus LABA and consider adding anti-IgE or anti-IL5 treatment.
- **Acute Exacerbations:** SABA for immediate relief, and systemic corticosteroids if no immediate improvement.
- **Asthma Action Plans:** Recommended for all patients to guide self-management.

Commonalities and Differences

- Both guidelines emphasize the use of short-acting beta-2 agonists for intermittent asthma and inhaled corticosteroids for persistent asthma.
- Both recommend step-up therapy based on the severity and control of asthma.
- GINA guidelines are more likely to recommend combination therapy (ICS + LABA) earlier in the treatment steps compared to NIH.

Medication Class	Specific Drugs	Mechanism of Action	Indications	Side Effects
<i>Long-Term Controller Medications</i>				

<i>Inhaled Corticosteroids (ICS)</i>	Fluticasone, Budesonide	Anti-inflammatory	Persistent asthma	Oral thrush, growth suppression First-line treatment
<i>Systemic Corticosteroids</i>	Prednisone, Prednisolone	Anti-inflammatory	Acute exacerbations	Weight gain, osteoporosis Monitor for side effects
<i>Long-Acting β-Agonists (LABA)</i>	Salmeterol, Formoterol	Bronchodilation	Add-on to ICS	Tachycardia, tremor Never use as monotherapy
<i>Combination ICS/LABA</i>	Fluticasone/Salmeterol	Anti-inflammatory + Bronchodilation	Moderate-to-severe asthma	Oral thrush, tachycardia Improved compliance
<i>Leukotriene Modifiers</i>	Montelukast, Zafirlukast	Block leukotriene pathway	Alternative to low-dose ICS	Liver function tests
<i>Long-Acting Anticholinergics</i>	Tiotropium	Bronchodilation	Add-on therapy (≥ 12 years)	Dry mouth, urinary retention
<i>Nonsteroidal Anti-Inflammatories</i>	Cromolyn Sodium, Nedocromil	Mast cell stabilization	Limited efficacy	Throat irritation
<i>Biologic Therapies</i>				
<i>Anti-IgE</i>	Omalizumab	Anti-IgE	Severe allergic asthma	Anaphylaxis
<i>Anti-IL-5</i>	Mepolizumab	Anti-IL-5	Severe eosinophilic asthma	Headache

Anti-IL-4 Receptor α	Dupilumab	Anti-IL-4 Receptor α	Moderate-to-severe asthma	Conjunctivitis
Quick-Reliever Medications				
Short-Acting β-Agonists (SABA)	Albuterol, Levalbuterol	Bronchodilation	Acute symptoms, exercise-induced bronchospasm	Tachycardia, tremor
Anticholinergic Agents	Ipratropium	Bronchodilation	Acute severe asthma	Dry mouth Usually in combination with SABA
Other Therapies				
Allergen Immunotherapy	Allergen-specific extracts	Sensitization reduction	Allergic asthma with known trigger	Local reactions, anaphylaxis

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Q: Management of status asthmaticus

Clinical manifestations of severe acute asthma:

Assess these in focussed history and physical examination

- PEFR < 4A% of predicted:
- Tachycardia > 120 beats/min:
- Tachypnoea > 40 breaths/min.
- Dyspnoea, inability to speak.
- Accessory muscle use
- Pulsus paradoxus > 15 mmHg.
- SaO₂ 90%-94%

Signs of Life-threatening attack

- Too dyspnoeic to speak.
- PEFR <25% predicted/patients best.
- Cyanosis

- Respiratory muscle fatigue- Abdominal paradox
- Altered consciousness -confusion, coma.
- A silent chest on auscultation.
- Pneumothorax
- Bradycardia, hypotension.
- SaO_2 , <90-92%, PO_2 <60 mmHg PaCO_2 > 45mmHg.

Management:

Phase I: One to one and a half hour

- O_2 by mask to keep O_2 saturation (SaO_2) > 90%.
- Start nebulisation with: Salbutamol 0.15-0.3 mg/kg/dose (max: 5 mg) and Budesonide (0.8 mg) per dose, upto 3 doses of at 20-30 min interval.
- Ipratropium 250 microgram every 30 minutes, up to 3 doses.
- Assess-Improved: Prepare for discharge / No/inadequate improvement: Follow phase II.

Phase II:

1. Continue oxygen.
2. Continue nebulised salbutamol—0.15 - 0.3 mg/kg/dose hrly or 0.3 mg/kg/hr continuous nebulization till response.
3. Set up IV line.
4. Systemic steroid -If conscious can accept orally—prednisolone 1 mg/kg/dose

If cannot take orally—Hydrocortisone 10 mg/kg stat then 5 mg/kg 6 hrly or methyl prednisolone 1-2 mg/kg/ dose IV. in 5% (D5) 500 ml + 2-8 mEq KCl infusion in 30 ml normal saline over 30 minutes.

5. Theophylline – After a bolus 5 mg/kg, 1mg/kg/hour by infusion.

It has beneficial therapeutic effect in children when given in combination with nebulized beta-agonists and systemic steroids.

6. Obtain blood counts, ABG's, Chest x-ray, electrolytes.

7. Treat infection if present with suitable antibiotics.

8. Reassessment—At end of 4 hours.

- Improved—Prepare for discharge.
- Inadequate/No response: Hospitalise, Follow phase III.

Phase III: Advanced Management of Status Asthmaticus in Children

- **Oxygen Therapy:** Continue oxygen administration to maintain adequate oxygen saturation.
- **Nebulized Salbutamol:** Continue either on a continuous or intermittent basis.
- **Magnesium Sulfate (MgSO₄) Infusion:** Continue 50% MgSO₄ infusions every 6 hours.
- **Intravenous Beta-Agonists:** Consider intravenous terbutaline or salbutamol for children who have not responded to nebulized bronchodilators and intravenous steroids.
- **Intravenous Salbutamol:**
 - Initiates a rapid decrease in PaCO₂ levels.

- Averts the need for mechanical ventilation in over two-thirds of cases.
- Dosing: Start at 0.5 µg/kg/min, increase by 1 µg/kg every 15 minutes until clinical improvement or PaCO₂ falls.
- Maximum infusion rate: 20 µg/kg/min, though most patients respond to 4 µg/kg/min.
- **Assisted Ventilation:**
 - Indicated if PaO₂ < 50 on 50% O₂, or PCO₂ > 50, or presence of cyanosis on 50% O₂.
- **Monitoring:**
 - Serial measurements of oxygen saturation.
 - Arterial blood gases (ABGs).
 - Cardiac and respiratory monitoring.

Ketamine:

- **Bronchodilatory Effects:** Ketamine has inherent bronchodilatory properties, making it a valuable adjunct in the management of severe status asthmaticus.
- **Sedative Properties:** As a dissociative anesthetic, ketamine provides sedation without compromising respiratory drive, which is crucial in severe asthma exacerbations.
- **Analgesic Effects:** Offers pain relief, which can be beneficial in the stressful situation of severe asthma exacerbation.
- **Synergy with Other Agents:** Can be used in conjunction with other bronchodilators and systemic corticosteroids to optimize airway management.
- **Dosing:** Typically administered as an intravenous infusion, starting at 0.2 to 0.5 mg/kg/hr, titrated to effect.

- **Cautions:** While generally well-tolerated, ketamine can cause increased secretions and hallucinations; thus, it should be used judiciously and typically in a controlled setting like the ICU.

Mechanical Ventilation:

- **Indications:**
 - Failure of medical management.
 - Deteriorating mental status.
 - Respiratory fatigue.
 - Severe hypoxia or hypercapnia.
- **Ventilation Modes:**
 - Volume-controlled or pressure-controlled modes are commonly used.
 - Permissive hypercapnia is often allowed to minimize barotrauma.
- **Ventilator Settings:**
 - Low tidal volumes (6–8 mL/kg of ideal body weight) to minimize the risk of volutrauma.
 - High inspiratory flow rates to decrease the inspiratory time, thus allowing more time for exhalation and reducing air trapping.
- **Monitoring:**
 - Continuous monitoring of hemodynamics, oxygenation, and ventilation parameters.
 - Frequent arterial blood gas analyses.
- **Weaning and Extubation:**

- Once the underlying severe asthma exacerbation has been controlled, and the patient meets extubation criteria, a weaning trial can be initiated.
- **Extracorporeal Membrane Oxygenation (ECMO):** For refractory cases.

Post-Stabilization

- **Reassessment:** Frequent clinical and ABG assessments.
- **Step-Down Therapy:** Gradual tapering of medications.
- **Education and Prevention:** Asthma action plan, trigger avoidance.

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Q: Clinical features, differential diagnosis, and treatment of atopic dermatitis in infant

Clinical Features of Atopic Dermatitis in Infants

- **Age of Onset:** Usually manifests within the first few months of life.
- **Distribution:** Commonly affects the cheeks, forehead, and extensor surfaces of the limbs.
- **Morphology:**
 - Erythematous patches or plaques
 - Vesicles and papules may be present
- **Pruritus:** Almost universally present, leading to scratching and potential secondary infection.
- **Chronicity:** Tends to be a chronic condition with periods of exacerbation and remission.
- **Family History:** Often a history of atopy in the family (asthma, allergic rhinitis, or atopic dermatitis).

Differential Diagnosis

- **Seborrheic Dermatitis:** Scalp involvement, less pruritus, and a greasier appearance.

- **Contact Dermatitis:** Triggered by specific allergens or irritants, localized to areas of contact.
- **Impetigo:** Presence of honey-colored crusts, usually caused by bacterial infection.
- **Scabies:** Intense pruritus, affecting other family members, and specific distribution such as finger webs.
- **Psoriasis:** Well-demarcated, scaly plaques; however, rare in infancy.

Treatment of Atopic Dermatitis in Infants

Topical Treatments

- **Emollients:** Liberal use of moisturizers to maintain skin hydration.
- **Topical Corticosteroids:** Low-potency corticosteroids like hydrocortisone for flare-ups.
- **Topical Calcineurin Inhibitors:** Tacrolimus or pimecrolimus for areas like the face where steroid use is risky.

Systemic Treatments

- **Antihistamines:** For symptomatic relief of pruritus, such as cetirizine.
- **Antibiotics:** Only if secondary bacterial infection is suspected or confirmed.

Environmental Controls

- **Allergen Avoidance:** Removal or minimization of known triggers.
- **Humidity Control:** Use of humidifiers in dry environments.

Dietary Management

- **Exclusive Breastfeeding:** Recommended for the first 6 months if possible.
- **Hypoallergenic Formulas:** For formula-fed infants, especially if a strong family history of atopy exists.

Monitoring and Follow-up

- **Regular Dermatological Assessment:** To assess the efficacy of treatment and make necessary adjustments.
- **Education:** Parental education on the chronic nature of the disease and proper skincare techniques.

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Q: A six-month old baby presents with rash over face and extensor aspect of extremities. Discuss the diagnosis and treatment of this baby

Diagnosis

Clinical Presentation

- **Age:** 6 months, which is a common age for the onset of atopic dermatitis.
- **Location of Rash:** Face and extensor aspects of extremities, typical sites for atopic dermatitis in infants.
- **Morphology:** If the rash appears as erythematous patches or plaques, possibly with vesicles or papules, it further supports the diagnosis.

Additional Diagnostic Steps

- **Family History:** Inquire about a family history of atopy (asthma, allergic rhinitis, or atopic dermatitis).
- **Pruritus:** If the infant seems uncomfortable and is scratching the affected areas, this is a strong indicator of atopic dermatitis.
- **Differential Diagnosis:** Rule out other conditions like seborrheic dermatitis, contact dermatitis, and impetigo through clinical features and possibly skin swabs or biopsies.

Treatment

Topical Management

- **Emollients:** Liberal application of moisturizers like petroleum jelly or ceramide-based creams to maintain skin barrier function.
- **Topical Corticosteroids:** A mild corticosteroid such as hydrocortisone 1% can be used for short periods to control flare-ups.

Systemic Management

- **Antihistamines:** A non-sedating antihistamine like cetirizine can be considered for symptomatic relief of itching.
- **Antibiotics:** Prescribe only if there is evidence of secondary bacterial infection, commonly caused by *Staphylococcus aureus*.

Environmental and Lifestyle Modifications

- **Allergen Avoidance:** Identify and remove potential allergens from the infant's environment, such as fragranced soaps or lotions.

- **Skin Care:** Use mild, fragrance-free cleansers and avoid hot baths.

Nutritional Considerations

- **Breastfeeding:** Continue breastfeeding if already doing so, as it may offer some protective effects.
- **Formula:** If formula-feeding, consider switching to a hypoallergenic formula.

Monitoring and Follow-up

- **Regular Assessments:** Schedule follow-up appointments to monitor the efficacy of the treatment regimen and adjust as necessary.
- **Parental Education:** Educate the parents about the chronic nature of atopic dermatitis and the importance of treatment adherence and regular follow-up.

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Differential Diagnosis	Clinical Features	Diagnostic Tests	Treatment	Remarks
Atopic Dermatitis	Erythematous, pruritic rash; commonly on face and extensor surfaces in infants. Dry skin.	Clinical diagnosis; may use patch testing to identify triggers.	Topical corticosteroids, emollients, antihistamines for itch.	Chronic, often familial; may be associated with other atopic conditions like asthma.

<i>Seborrheic Dermatitis</i>	Greasy, yellowish scales on scalp and face; may extend to diaper area.	Clinical diagnosis; KOH test to rule out fungal infection.	Topical antifungals or low-potency corticosteroids.	Common in infants; tends to resolve by 6-12 months.
<i>Contact Dermatitis</i>	Erythematous, pruritic rash at sites of contact with irritants or allergens.	Patch testing for allergens.	Removal of irritant/allergen; topical corticosteroids.	History of exposure to new products is common.
<i>Impetigo</i>	Honey-colored crusts, often around the mouth and nose; can be pruritic.	Culture of lesion.	Topical or systemic antibiotics depending on severity.	Highly contagious; common in children.
<i>Scabies</i>	Intensely pruritic rash, often with burrows; commonly affects webs of fingers and wrists.	Microscopic examination of skin scrapings.	Topical or oral scabicides.	Close contact history often present.
<i>Psoriasis</i>	Well-demarcated, erythematous plaques with silvery scales; rare in infants.	Skin biopsy.	Topical corticosteroids; UV therapy in severe cases.	Chronic condition; may have family history.
<i>Urticaria (Hives)</i>	Transient, pruritic, raised wheals; can appear and disappear rapidly.	Clinical diagnosis; allergy testing.	Antihistamines; corticosteroids for severe cases.	Often triggered by allergens or infections.
<i>Infantile Hemangioma</i>	Red or bluish raised lesion; commonly on head and neck.	Clinical diagnosis; ultrasound for deeper lesions.	Observation; propranolol for problematic lesions.	Benign; often resolves spontaneously.

Eczema Herpeticum	Vesicular rash with erosions; can be a complication of atopic dermatitis.	Tzanck smear; viral culture.	Systemic antivirals.	Requires prompt treatment to prevent complications.
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Q: Treatment for Urticaria

• **First-Line Therapy**

- Antihistamines: Cetirizine, Fexofenadine, Loratadine
- Mechanism: Block H1 receptors, reducing histamine-induced vasodilation and pruritus.
- Dosing: Varies by agent; generally once daily.

• **Second-Line Therapy**

- H2 Antagonists: Ranitidine, Famotidine
- Mechanism: Block H2 receptors, providing additional control of histamine-mediated symptoms.
- Dosing: Varies by agent; generally once or twice daily.

• **Short-Term Symptomatic Relief**

- Corticosteroids: Prednisone
- Mechanism: Anti-inflammatory; suppress immune response.
- Dosing: Short course, typically less than 5 days to minimize side effects.

• **Chronic Cases**

- Leukotriene Receptor Antagonists: Montelukast
- Mechanism: Block leukotriene receptors, reducing inflammation.
- Dosing: Once daily, usually in the evening.

• **Severe or Refractory Cases**

- Omalizumab: Anti-IgE monoclonal antibody
- Mechanism: Binds to IgE, preventing it from attaching to mast cells and basophils.

- Dosing: Subcutaneous injection every 2-4 weeks.
- **Adjunctive Therapies**
 - Epinephrine: For anaphylactic reactions
 - Mechanism: Alpha and beta-adrenergic agonist; reverses airway obstruction and improves hemodynamics.
 - Dosing: Single intramuscular injection; repeat as needed.
- **Non-Pharmacological Interventions**
 - Avoidance of Triggers: Identification and avoidance of allergens or irritants.
 - Cold Compress: To reduce local inflammation and pruritus.
- **Monitoring and Follow-Up**
 - Regular assessment for efficacy and side effects of treatment.
 - Adjustment of treatment regimen based on symptom control.

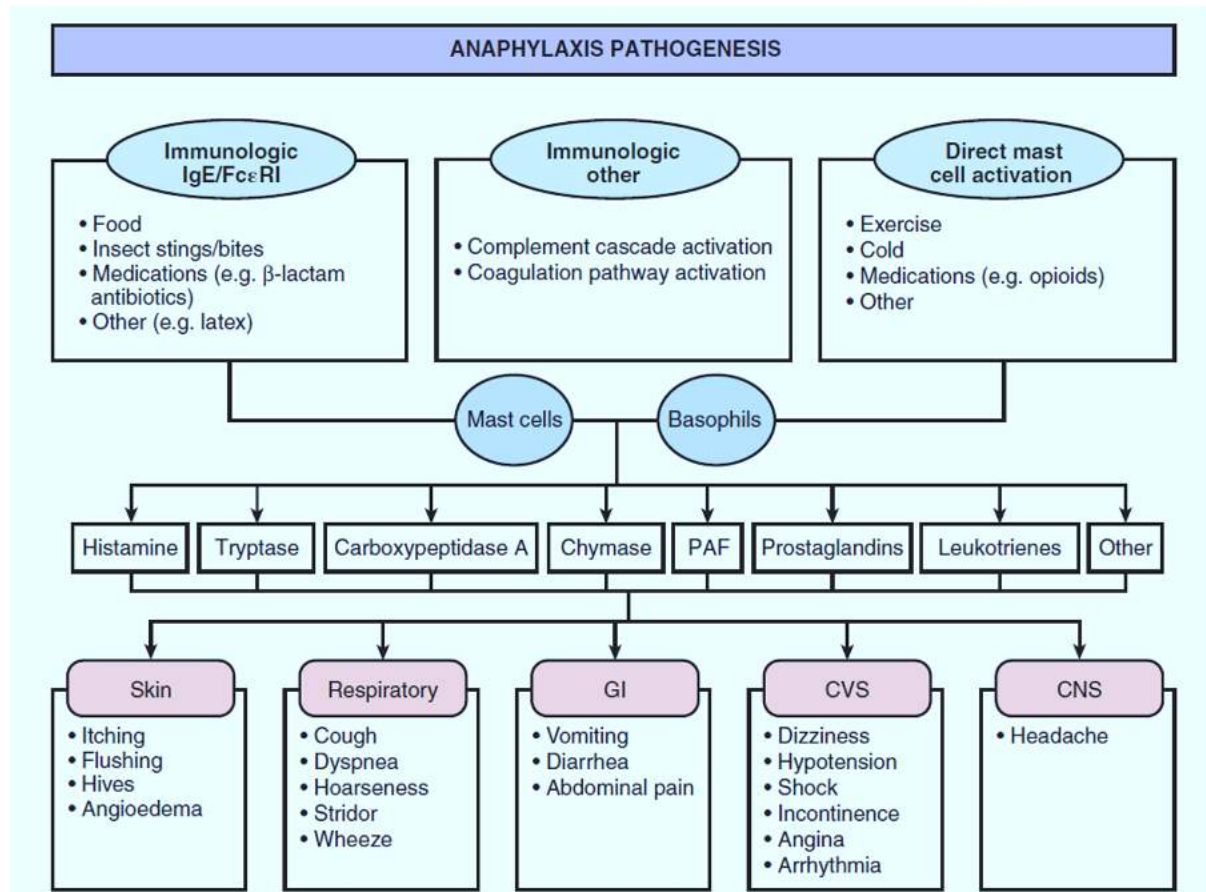
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Q: Pathogenesis and management of anaphylaxis

Pathogenesis of Anaphylaxis

- **IgE-Mediated Mechanism**
 - Allergen exposure triggers IgE antibodies to bind to mast cells and basophils.
 - Subsequent allergen exposure cross-links IgE, leading to degranulation of mast cells and basophils.
 - Release of histamine, leukotrienes, and prostaglandins.
- **Non-IgE-Mediated Mechanism**
 - Complement activation, direct mast cell activation, or bradykinin pathways.
 - Examples include reactions to radiocontrast media, opioids, and certain foods.
- **Mixed Mechanisms**
 - Both IgE and non-IgE pathways involved.

- Examples include some drug allergies and idiopathic anaphylaxis.
- **Cytokine Storm**
 - Overactivation of immune cells leading to excessive cytokine release.
 - Seen in severe cases and can lead to multi-organ failure.
- **Direct Mast Cell Activation:**
- There are certain triggers that can directly activate mast cells, bypassing typical allergic pathways.
- These triggers include:
 - **Exercise**
 - **Cold**
 - **Medications:** E.g., opioids
 - **Other:** Non-specified triggers
- Regardless of the initiating pathway, the activation culminates in the release of mediators from **mast cells** and **basophils**. These mediators include:
 - **Histamine**
 - **Tryptase**
 - **Carboxypeptidase A**
 - **Chymase**
 - **PAF (Platelet-activating factor)**
 - **Prostaglandins**
 - **Leukotrienes**
 - **Other non-specified mediators**



Management of Anaphylaxis

• Immediate Interventions

- Epinephrine: Intramuscular injection, 0.01 mg/kg, up to 0.3 mg for adults.
- Oxygen: To maintain SpO₂ > 94%.
- Intravenous Fluids: Crystalloids for hypotension.

• Adjunctive Medications

- Antihistamines: Diphenhydramine or cetirizine for cutaneous symptoms.
- Corticosteroids: Methylprednisolone or hydrocortisone to prevent **biphasic anaphylaxis**.
- Bronchodilators: Albuterol for bronchospasm if not relieved by epinephrine.

• Monitoring

- Continuous monitoring of vital signs, cardiac rhythm, and oxygen saturation.

- Serial measurement of tryptase levels for diagnosis confirmation.
- **Supportive Measures**
 - Airway management: Endotracheal intubation or surgical airway for severe respiratory distress.
 - Vasopressors: Norepinephrine or dopamine for refractory hypotension.
- **Post-Emergency Management**
 - Observation: At least 4-6 hours in a healthcare facility.
 - Anaphylaxis action plan and epinephrine auto-injector prescription.
 - Referral to an allergist for identification of triggers and desensitization.
- **Long-Term Management**
 - Allergen avoidance and education.
 - Regular follow-up with an allergist.

Q: Cow's Milk Allergy (CMA);

- **Definition:** Immunologically mediated adverse reaction to one or more cow's milk proteins.
- **Onset:** Usually within the first year of life.
- **Immunological Mechanisms:**
 - IgE-mediated (Type I hypersensitivity)
 - Non-IgE-mediated (Type IV hypersensitivity)
 - Mixed
- **Clinical Manifestations:**
 - Cutaneous: Urticaria, eczema, angioedema.
 - Gastrointestinal: Vomiting, diarrhea, bloody stools.
 - Respiratory: Wheezing, cough, rhinitis.
 - Systemic: Anaphylaxis.
- **Diagnosis:**
 - Clinical history and physical examination.

- Skin prick test or serum-specific IgE testing.
- Elimination diet followed by oral food challenge.
- **Management:**
 - Strict avoidance of cow's milk and its derivatives.
 - Use of hypoallergenic formulas.
 - Epinephrine auto-injector for severe cases.
- **Cow's Milk Protein Intolerance (CMPI)**
 - **Definition:** Non-immunological adverse reaction to cow's milk proteins.
 - **Onset:** Variable, can occur at any age but commonly seen in infants.
 - **Mechanisms:**
 - Metabolic (e.g., lactose intolerance)
 - Pharmacological (e.g., reactions to amines in foods)
 - Undefined
 - **Clinical Manifestations:**
 - Gastrointestinal: Colic, reflux, diarrhea, constipation.
 - No risk of anaphylaxis.
 - **Diagnosis:**
 - Clinical history and physical examination.
 - Elimination diet followed by oral food challenge.
 - No reliable laboratory tests.
 - **Management:**
 - Dietary modification, often with the use of specialized formulas.
 - Lactase supplements for lactose intolerance.
- **Differential Features**
 - **Immunological Basis:** Present in CMA, absent in CMPI.
 - **Risk of Anaphylaxis:** Present in CMA, absent in CMPI.
 - **Diagnostic Tests:** More definitive in CMA.
 - **Treatment:** More stringent avoidance in CMA.

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Q: Immunological features associated with cow milk allergy

- **Type I Hypersensitivity**
 - Immediate reaction mediated by IgE antibodies.
 - Mast cell and basophil degranulation releasing histamine and other mediators.
 - Clinical manifestations: Urticaria, angioedema, anaphylaxis.
- **Type III Hypersensitivity**
 - Immune complex-mediated.
 - Formation of antigen-antibody complexes that deposit in tissues.
 - Clinical manifestations: Serum sickness-like reactions.
- **Type IV Hypersensitivity**
 - T-cell mediated, delayed-type hypersensitivity.
 - Activation of Th1 and Th17 cells.
 - Clinical manifestations: Atopic dermatitis, contact dermatitis.
- **Non-IgE Mediated Gastrointestinal Allergies**
 - Mechanism not fully understood but involves T-cell activation.
 - Clinical manifestations: Food protein-induced enterocolitis syndrome (FPIES), allergic proctocolitis.
- **Mixed Mechanisms**
 - Both IgE and T-cell mediated.
 - Clinical manifestations: Eosinophilic esophagitis.
- **Sensitization**
 - Presence of specific IgE antibodies against cow's milk proteins in the blood.
 - Not all sensitized individuals show clinical symptoms.
- **Cross-Reactivity**
 - IgE antibodies against cow's milk proteins may cross-react with proteins in goat's and sheep's milk.

- **Tolerance Development**

- Some children may develop tolerance over time.
- Mechanisms include the development of regulatory T cells and changes in IgE affinity.

- **Biomarkers**

- Elevated levels of serum tryptase, eosinophilia.
- Elevated levels of fecal calprotectin in non-IgE mediated cases.

- **Genetic Predisposition**

- Higher risk in individuals with a family history of atopy.

- **Skin Testing**

- Positive skin prick test or atopy patch test for cow's milk proteins.

- **Cytokine Profile**

- Elevated levels of pro-inflammatory cytokines like IL-4, IL-5, and IL-13 in affected tissues.

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Q: Clinical features and management of Atopic dermatitis

- ❖ Atopic dermatitis, also known as eczema, is a chronic, inflammatory skin condition characterized by dry, itchy skin.
- ❖ It's commonly seen in children but can occur at any age. Atopic dermatitis requires a multifaceted approach to management, focusing on skin care, avoidance of triggers, and appropriate use of medications.
- ❖ Regular follow-up and patient education are key components in managing this chronic condition effectively.

Clinical Features

1. **Age of Onset:** Often begins in infancy or early childhood.
2. **Itching:** The hallmark symptom, often severe and worse at night.
3. **Skin Changes:** Dry, scaly, and thickened skin; acute lesions may be weepy and crusted.
4. **Distribution:** Typically affects the face, scalp, and extensor surfaces in infants; flexural areas (inside elbows, behind knees) in older children and adults.

5. **Chronic Course:** Characterized by flare-ups and remissions.
6. **Atopic Triad:** Often associated with asthma and allergic rhinitis.
7. **Skin Infections:** Increased susceptibility to bacterial (e.g., *Staphylococcus aureus*) and viral (e.g., herpes simplex) infections.

Management

Non-Pharmacological

1. **Skin Care:** Regular use of moisturizers to maintain skin hydration.
2. **Avoid Triggers:** Such as irritants (wool, harsh soaps), allergens, and stress.
3. **Bathing Practices:** Short, lukewarm baths with mild soap, followed by immediate moisturizing.
4. **Nail Care:** Keeping nails short to minimize skin damage from scratching.
5. **Humidifiers:** In dry climates, to keep air moist and prevent skin dryness.

Pharmacological

1. **Topical Corticosteroids:** First-line treatment for flare-ups, with potency tailored to the severity of symptoms and age of the patient.
2. **Topical Calcineurin Inhibitors:** Such as tacrolimus and pimecrolimus, for areas where steroid use is limited (e.g., face, neck).
3. **Systemic Therapy:** For severe cases, options include oral corticosteroids, cyclosporine, methotrexate, or dupilumab.
4. **Antihistamines:** Primarily for itch relief, especially sedating types at night to aid sleep.
5. **Antibiotics:** For secondary bacterial infections.
6. **Wet Wrap Therapy:** For severe flare-ups, involving the application of wet bandages over topical medications to enhance their effect and hydrate the skin.

Education and Support

1. **Patient Education:** About the chronic nature of the disease and the importance of regular skin care.
2. **Psychological Support:** For patients and families dealing with the stress and impact of the disease.

3. **Dietary Considerations:** In some cases, dietary triggers may need to be identified and avoided.

Monitoring and Follow-Up

1. **Regular Assessments:** To monitor the effectiveness of treatment and adjust as needed.
2. **Skin Infection Signs:** Educating patients on recognizing signs of infection and seeking prompt treatment.

Complications

1. **Eczema Herpeticum:** A serious complication due to herpes simplex virus infection.
2. **Eye Complications:** Including conjunctivitis, keratitis, and cataracts.
3. **Psychosocial Impact:** Including sleep disturbance and impact on quality of life.

The logo for My Doctor Guru, featuring a circular emblem with a caduceus and the text 'My Doctor Guru' in a stylized font.

Q: Clinical evaluation of severity of acute exacerbation of bronchial asthma in children

The clinical evaluation of the severity of an acute exacerbation of bronchial asthma in children involves a comprehensive assessment of various signs and symptoms, as well as the child's response to initial treatment.

Initial Assessment

1. **History of Present Illness:** Duration, triggers, and progression of symptoms.
2. **Medication History:** Current asthma medications and adherence.
3. **Previous Asthma Exacerbations:** Frequency, severity, and any previous hospitalizations or intensive care admissions.

Physical Examination

1. **General Appearance:** Assess for signs of distress, ability to speak or cry, and level of consciousness.
2. **Respiratory Rate:** Increased rate indicative of respiratory distress.
3. **Heart Rate:** Tachycardia often accompanies severe exacerbations.

4. **Oxygen Saturation:** Measured by pulse oximetry; levels below 92% in room air are concerning.
5. **Use of Accessory Muscles:** Intercostal, subcostal, or supraclavicular retractions.
6. **Auscultation:** Presence and extent of wheezing, diminished breath sounds, or silent chest (a severe sign indicating minimal air movement). Pulsus paradoxus on BP measurement.
7. **Cough:** Frequency and character.
8. **Ability to Talk or Feed:** Difficulty speaking or feeding indicates severe distress.

Severity Classification

1. **Mild Exacerbation:** Increased cough and wheeze, but no difficulty with feeding or talking, normal oxygen saturation, and no or minimal use of accessory muscles.
 2. **Moderate Exacerbation:** More pronounced symptoms, mild to moderate distress, may have slightly decreased oxygen saturation, and increased use of accessory muscles.
 3. **Severe Exacerbation:** Marked respiratory distress, difficulty speaking or feeding, use of accessory muscles, and potentially decreased oxygen saturation.
 4. **Life-Threatening Exacerbation:** Extreme difficulty breathing, altered level of consciousness, cyanosis, silent chest on auscultation, and/or oxygen saturation persistently below 92%.
- The below scoring systems are used to assess the severity of asthma exacerbations in pediatric patients.
 - They help in guiding treatment decisions and evaluating the effectiveness of interventions.

Pediatric Asthma Severity Score (PASS)

Component	Scoring	Criteria
Respiratory Rate	0-3	Scored based on age-specific normal ranges
Oxygen Saturation	0-2	>95% = 0, 92-95% = 1, <92% = 2

<i>Auscultation (Wheeze)</i>	0-3	None = 0, End-expiratory = 1, Throughout expiration = 2, Inspiratory and expiratory = 3
<i>Work of Breathing</i>	0-4	Normal = 0, Mild (intercostal retractions) = 1, Moderate (intercostal and substernal retractions) = 2, Severe (above plus nasal flaring and/or tracheal tugging) = 3, Agonal breathing = 4

Interpretation:

- Total Score: 0-12
- 0-4: Mild Asthma
- 5-8: Moderate Asthma
- 9-12: Severe Asthma

Pulmonary Index Score (PIS)

Component	Scoring	Criteria
<i>Respiratory Rate</i>	0-3	Scored based on deviation from normal for age
<i>Wheezing</i>	0-3	None = 0, End-expiratory = 1, Throughout expiration = 2, Inspiratory and expiratory = 3
<i>Inspiratory/Expiratory Ratio</i>	0-3	Normal = 0, Mild prolongation of expiration = 1, Moderate prolongation = 2, Severe (inspiration = expiration) = 3
<i>Accessory Muscle Use</i>	0-3	None = 0, Mild = 1, Moderate = 2, Severe = 3
<i>Dyspnea</i>	0-3	None = 0, Mild = 1, Moderate = 2, Severe = 3

Interpretation:

- Total Score: 0-15
- 0-5: Mild Asthma
- 6-10: Moderate Asthma
- 11-15: Severe Asthma

Additional Investigations

1. **Peak Expiratory Flow Rate (PEFR):** Reduced in exacerbations, but may be difficult to measure in young children.
2. **Chest X-Ray:** To rule out complications like pneumothorax or pneumonia.
3. **Blood Gases:** In severe cases, to assess for hypoxemia or hypercapnia.
4. **Complete Blood Count (CBC):** To rule out infection if indicated.

Response to Initial Treatment

1. **Bronchodilator Response:** Improvement or lack thereof after initial nebulized bronchodilator treatment.
2. **Systemic Corticosteroids:** Assess response to oral or intravenous corticosteroids.

Monitoring

1. **Continuous Monitoring:** Of oxygen saturation and respiratory status.
2. **Frequent Reassessment:** To monitor response to treatment and progression of symptoms.

Decision Making

1. **Hospitalization Criteria:** Severe exacerbations, poor response to initial treatment, or significant distress.
2. **Intensive Care Consideration:** For life-threatening exacerbations or if there's a risk of respiratory failure.

